ON A

CASE OF PATENT DUCTUS ARTERIOSUS

WITH

ANEURISM OF THE PULMONARY ARTERY.

BY

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ON A

CASE OF PATENT DUCTUS ARTERIOSUS.

Mr President and Gentlemen,—One morning, early in the year 1882, a young girl came to my consulting-room for advice for the following distressing symptoms:—Great palpitation of the heart on slight exertion, breathlessness, and buzzing noises in her chest, in her head and ears. She was very nervous, and her face bore an extremely anxious expression. The nose and lips were pale, with a slightly bluish tinge, and the conjunctival mucous membranes were very anæmic and flabby. There was no cough. Concluding from her pale face and lips that her symptoms were in a great measure due to bloodlessness, I made a very superficial examination at this time; but on placing the stethoscope over the base of the heart, I heard a loud blowing murmur, and at the same time felt a strong heaving impulse accompany the heart's systole. Immediately following the systolic murmur, another softer murmur was heard. These extraordinary murmurs puzzled me very much, as they were not at all like the ordinary humming murmurs of anæmia. I at once prescribed iron and arsenic and rest, and obtained her promise that she would come back to me in three weeks time for further examination and advice, hoping by that time that her bloodlessness would have disappeared, and I should be better able to localize the murmurs and discover the nature of her cardiac affection. On her reappearance in three weeks time her face and lips showed that she had gained much blood, and that her general health had greatly improved; but on examining her heart at its base, to my intense surprise, I heard the murmurs louder than they were before, and much more marked in every respect. Her pulse was fairly good and quite regular. She still complained of palpitation on exertion, and her dyspnœa was little, if anything better; also the noises in her head and chest distressed her very much. On placing the stethoscope over the base of the heart, to the left side of the sternum, in the region of the second intercostal space, two loud murmurs were heard. The first murmur, which was

very loud and blowing with a decided thrill in it, accompanied the systole of the heart, and at the same time a strong heaving impulse was communicated to the stethoscope. Just before this murmur ceased a distinct "click" was heard, and then immediately a second murmur, but of a softer nature and of shorter duration than the first murmur, was heard. On moving the stethoscope to the aortic area, the murmurs were still very distinctly heard, but the click which, as I have said, joined the two murmurs together was heard much more clearly, and I believe indicated the quick and sudden closure of the aortic valve. How the murmurs themselves were produced I could not at this time venture to say. They were heard also at the apex of the heart, but not nearly so loud as at the spot already stated. *The point of maximum intensity of both murmurs was to the left of the sternum between the second and third ribs.*

Upon entering into the history of my patient, I learned that about six years previously she had an attack of scarlet fever, but she could not say that her health in any way was worse after the attack. There was no history of rheumatic affection. I advised my patient to give up all unnecessary exertion, and to rest as much as possible, and to continue taking the iron and arsenic for another month.

At the end of a month I again saw her. She was looking greatly better. Her face and lips showed that her anæmia had quite disappeared. She still had great breathlessness on exertion, and complained much of the noises in her head and chest.

On listening over the base of the heart to the left of the sternum, in the second intercostal space, a strong heaving impulse was communicated to the stethoscope with the systole of the heart, and both murmurs, as already described, were heard with extraordinary clearness—I never heard anything like those murmurs before or since. At the apex of the heart the murmurs were not so loud as at the spot indicated; and in the aortic area, while the click joining the two murmurs was very distinct, the murmurs themselves were but slightly diminished in intensity.

Being very much puzzled as to the cause of these murmurs, which, be it borne in mind, were much louder and better marked since the girl's anæmia had disappeared, I thought it would be interesting to hear what Dr George Balfour had to say as to the cause of the murmurs. After a careful examination he came to the conclusion that the case was one of patent ductus arteriosus, but at the same time confessed that his diagnosis was founded upon hypothetical rather than well-known scientific data. As regards treatment of the case, Dr Balfour advised that she should continue to take iron and arsenic, and should rest as much as possible.

From time to time I visited my patient at her own home, as she was unable to come to my house. At this time my friend Dr Wyllie saw her with me. He also kindly took me to see a case

in the Infirmary which was of great interest to the physicians. The case was believed to be one of patent ductus arteriosus. I had an opportunity of examining this girl immediately after seeing my own patient. On auscultation in the second left intercostal space two loud murmurs were heard very like those described as existing in my own case, but both murmurs seemed coarser and rougher, and the girl herself had a very healthy, even ruddy appearance.

The summer being at hand, my patient went into the country for change of air, and I did not see her for two or three months, but I heard from a medical friend that he had been called to treat my patient for hæmoptysis and bleeding at the nose. From this time her health began to fall off greatly; she suffered much from cough. Her feet were occasionally swollen, and she became very anæmic.

I heard from the girl's mother frequently to this effect,—that my patient suffered greatly from breathlessness and cough, and that she often coughed up some blood, and had had several attacks of bleeding at the nose, and was now extremely pale and weak. As she was rapidly getting worse, I advised her return to Edinburgh so as to be under my care.

She returned to Edinburgh on the afternoon of the 31st August, and I saw her at 8.30 P.M. She was then sitting up in a chair by the fireside looking extremely pale and ill. What struck me at once was the quickness of her breathing. Her pulse was 120, and respirations about 54 per minute. On examining the chest there were crackling, moist sounds on both sides in front and behind; there was constant cough, with some frothy and bloody expectoration. The veins in the neck were much distended. She could not lie down, but was kept in bed propped up with pillows. On placing the stethoscope over the base of the heart on the second intercostal space, a very strong heaving impulse was communicated to the instrument, but, to my great surprise, both loud murmurs had almost entirely disappeared. The heart was violently wobbling about and striking against a large area of the chest wall, and occasionally I heard two sounds which I can best describe by the words "filupp," "flupp," pronounced quickly. How these sounds were produced I could not then say, but I came to the conclusion that all the cavities of the heart were extremely overgorged and in a state of tension.

The pallid face and bloodless and bluish lips showed that she was extremely anæmic and somewhat cyanotic. Day by day she lost more blood in the frothy expectoration. Her urine, which was very scanty, contained one-fifth albumen. She was at times delirious. She was not allowed to take any food but milk and water with white of egg. For some time she showed a decided improvement, but the distressing cough, with bloody expectoration, never left her. Some days better, some days worse, she lingered

on until about the 20th September, when she began to complain of sharp cutting pain in the region of her heart; and on stethoscopic examination a loud, continuous, rough friction sound was heard all over the cardiac area. Pericarditis had evidently set in. After her return from the country I did not again hear the remarkable murmurs. They had entirely disappeared; but it was often possible to hear the "filupp," "flupp" sounds during the violently wobbling action of the heart. She died on the 25th September, thoroughly worn out and exhausted, 22 years of age.

Such is the very imperfect history of this case so far, but I am indebted to my friend Dr Wyllie for the following notes taken at his dictation at the post-mortem examination performed on the 27th September 1882:—

Description of Body.—The body was 5 ft. $1\frac{1}{4}$ inches in length. Circumference of body just below the level of the mammæ was $27\frac{3}{4}$ inches. The body was delicately formed, rather emaciated. Mammæ rather small. There were evidences of commencing putrefaction, abdomen being distended and tympanitic. Skin over the abdominal region being greenish blue in tint. The same tint of skin was seen on the throat. Rigor mortis still slightly present in the legs, but absent in neck and upper extremities. The legs below the knees were somewhat œdematous, pitting on pressure being best marked above the ankles.

On proceeding to examine the chest, the skin over the costal parietes was first reflected before the thoracic or abdominal cavities were opened into. The chest wall was then transfixed with four long barbed crotchet needles at the following points:—

Needle No. 1 was driven into the second left intercostal space, exactly at middle distance between the two cartilages, at a spot one inch to the left of the sternal margin.

Needle No. 2 was inserted in the same intercostal space exactly between the two ribs, but at the distance of $\frac{1}{2}$ inch from the left margin of the sternum.

Needle No. 3 was inserted into the third left intercostal space, exactly at mid distance between the third and fourth ribs, at a spot $\frac{1}{2}$ inch from the left margin of the sternum.

Needle No. 4 was inserted in the second *right* intercostal space, mid way between the ribs, at a spot $\frac{1}{2}$ inch from the right margin of the sternum.

These needles, being thus fixed in position, were driven deeply inwards in a vertical direction, and the costal cartilages were divided in the usual way on each side, and the sternum with the attached cartilages and soft parts was removed. The needles, being fixed by their barbed points to the cardiac tissues, were left attached to the heart after the sternum had been removed.

On thus removing the sternum the needles which had been inserted to the left side of the sternal bone were all found to enter directly into the anterior surface of the pericardial sac without

piercing the margin of the left lung, which lay close, ¼ inch, to the outer side of needle No. 1. On the other hand, the needle No. 4, which was inserted half an inch to the right of the sternum in the second intercostal space, transfixed the margin of the right lung in its upper lobe ¾ inch to the right of its free edge. Having thus transfixed the lung, this needle passed into the surface of the pericardium close to the right margin of the sac.

The *pericardium* was next opened. The sac was found to contain about ½ pint of blood-stained serum, and the serous membrane, parietal and visceral, was found to be coated with a delicate villous layer of soft and recent lymph, which extended in patches over the whole surface of the heart and its containing sac.

The following was now found to be the positions of the needles:—

Needle No. 1 pierced the anterior surface of the pulmonary artery about its middle, and at a spot about ¼ inch above its valve.

Needle No. 2 transfixed the *aorta* immediately above its valve, passing immediately to the right of the pulmonary artery before piercing the aorta.

Needle No. 3 pierced the conus arteriosus 1 inch below the pulmonary valve, and at a spot ¼ inch to the right of the middle line of the conus arteriosus, and then passed backwards through the interventricular septum, and transfixed the left ventricle about an inch below the aortic valve.

Needle No. 4 passed through the pericardial sac at its outer limit, and merely transfixed a portion of the right wall of the heart without entering its chambers.

The Heart as a whole was much enlarged, measuring in length from pulmonary valve to apex 4¾ inches; and when flattened out, it measured in breadth across its middle 4½ inches. Its muscular substance was a good deal softened by putrefaction. There was evident hypertrophy of both ventricles, the left wall measuring ⅝ inch in thickness, and the right wall measuring ⅜ inch in thickness. The ventricular cavities were both considerably dilated. The auricles were not pierced by any of the needles. The left auricle was only slightly dilated, and its appendix was empty and flaccid, being in position behind and to the left of the pulmonary artery, and quite overshadowed by the aneurism on the pulmonary artery to be presently described. The appendix of the left auricle as a whole was large enough to contain easily the point of the forefinger, but the cavities of its fringe-like margins were not large enough to permit the entrance of the little finger point. The right auricle was considerably dilated, except as regards its appendix, which was of natural size.

The Lungs.—The right lung, especially at its upper lobe, contained a number of old and indurated infarctions of a grayish red colour. Both lungs were greatly congested and œdematous, with a feeling of partial consolidation, probably the result of catarrhal pneumonia.

The Liver was natural.

Spleen was greatly enlarged, measuring 8 inches in length by $4\frac{1}{2}$ in breadth.

The Kidneys were much congested and enlarged, and were softened by putrefaction; otherwise they were natural.

The heart, pericardium, and great vessels, were now removed and carefully dissected, without severing the connexion of the heart and its great vessels.

The following was found to be the condition of the great vessels:—

The Conus Arteriosus was very considerably dilated. When the pericardium was first opened its anterior surface was found bulging forward with unnatural prominence.

The Pulmonary Artery measured from the base of its valvular cusps to its bifurcation, $2\frac{5}{8}$ inches. At the bifurcation the ductus arteriosus communication with the aorta was patent; the orifice of communication being large enough to admit freely a goose quill.

The Ductus Arteriosus was represented by a very short vascular trunk about $\frac{1}{3}$ inch in length. When viewed externally its narrowest diameter appeared to be about $\frac{1}{4}$ inch. At its aortic attachment there was a bulging of the aortic wall, which corresponded with the funnel-shaped opening of the ductus arteriosus on the inner wall of the aorta.

The Pulmonary Artery, as a whole, was found to be dilated to at least twice its natural size, but this dilatation was not uniform in all directions, for whilst the posterior, right lateral, and anterior walls presented no special bulging or marked irregularity of outline, the left lateral wall from the ductus arteriosus close down to situation of the pulmonary valve was bulged in an outward direction towards the left, and at the lower limit, where the bulging was greatest, a regular aneurismal sac was formed about the size of a large walnut. This sac was contained within the pericardium, and, as already said, lay in front of the left auricular appendix, which it entirely hid from view.

The needle No. 1 transfixed the middle of the pulmonary artery, about $\frac{3}{4}$ inch to the right of the aneurismal sac. The sac was filled internally with pretty firm coagulum, and from the sac this coagulum extended along the surface of the left anterior wall of the artery to near its bifurcation, being bound to this wall by pretty firm adhesion. A conical prolongation of this clot partially blocked the lumen of the ductus arteriosus. Externally, the part of the sac which was contained within the pericardium was covered with abundant inflammatory lymph of older date than the lymph effused on the pericardial surface generally. This lymph was deeply stained with blood colouring matter, and formed firm adhesions between the aneurismal sac and the parietal layer of the pericardium.

The primary divisions of the pulmonary artery were both of large size, the right being at least nearly twice as large as usual.

Into the left division there projected for about half an inch an offshoot of the coagulum above described as existing in the main trunk of the pulmonary artery. This, however, occupied only about one-fourth or one-sixth of the lumen of the vessel.

The Valves of the Heart.

The Pulmonary Valve was extensively diseased. Each cusp on its ventricular surface was covered with ragged vegetations, attached for the most part to the neighbourhood of the corpus Arantii and to the margin and upper half of the cusp. These vegetations were of soft granular consistence, and appeared to be made up in part of true vegetative growths and in part of secondary fibrinous deposit. The anterior cusp was the least diseased, retaining its full size, but presenting a large amount of vegetations in the neighbourhood of the corpus Arantii. The two posterior cusps, right and left, were both reduced to about half their natural dimensions by ulceration or atrophic change, and were fringed along the whole length of their free margins with large and ragged vegetations of soft consistence. The ragged and vegetating edges of these two cusps were both turned over so as to hang as it were dependent towards the ventricular cavity, and in the case of the anterior cusp it was evident from the dipping of its free margin that there had been a tendency to a similar state of matters.

The circumference of the pulmonary orifice at the level of its cusps was $3\frac{1}{2}$ inches.

The Aortic Valve.—The orifice of the aorta was about natural in size. All the cusps of the aortic valve, however, presented on their ventricular surface vegetations attached to the corpora Arantii. In the case of the anterior cusp the group of vegetations formed a mass the size of a small pea. On the other cusps the vegetations were much more minute, being about the size of small pin heads. The cusps otherwise retained their natural size and structure.

The Mitral Valve.—The anterior or aortic cusp was well developed, and was natural, except that on its auricular surface, close to its free margin, it presented an attached mass of dense vegetation about the size of a small bean.

The Aorta was of natural size, both in its arch and descending portion. At the junction of the second and third portions of the arch, external to the line of the left subclavian artery, was the patent opening of the ductus arteriosus already referred to. The margins of the opening were smooth, and the wall of the aorta at the point of junction with the ductus arteriosus was distended somewhat in a funnel-like manner. The lumen of the ductus arteriosus was large enough to transmit a goose quill, and was partly plugged by a protruding offshoot of the coagulum from the pulmonary artery, as already said. On the posterior wall of the

aorta, *exactly opposite the orifice of the patent ductus arteriosus,* there was a rounded bulging or lens-shaped concavity in the aortic wall, which measured one inch in diameter, and in depth about half an inch. The cavity thus formed was partly filled up with adherent coagulum, and the limiting wall of the cavity was here hardened as if by atheromatous changes.

The Mitral Orifice was slightly dilated.

The Tricuspid Orifice was almost natural in size, and its cusps were quite normal.

The Foramen Ovale was quite closed.

The same letters when present in both Plates point to the same parts.

Plate I.

Fig. 1.—Shows the heart with its large bloodvessels. All the cavities of the heart and the large bloodvessels were stuffed with wet cotton wool, so as to distend them. In this way the dilated right ventricle, conus arteriosus, and pulmonary artery are well shown. The descending portion of the aorta has been raised up from its normal position, so as to bring more clearly into view the ductus arteriosus.

A, The innominate artery.
B, Left carotid artery.
C, Left subclavian artery.
D, The patent ductus arteriosus.
E, Left branch of the pulmonary artery.
F, Descending portion of the aorta.
G, Saccular aneurism, filled with granular clot, on the left anterior wall of the pulmonary artery.
H, Dilated left ventricle.
I, Dilated right ventricle.
K, Right auricle.
L, Ascending aorta.
M, This letter is placed below the needle No. 1, which was inserted into the second left intercostal space, midway between the ribs, at a spot 1 inch to the left of the sternal margin. It pierced the pulmonary artery at its middle, about ¼ inch above its valve.
N, This letter is placed below the needle No. 3, which was inserted into the third left intercostal space, midway between the ribs, at a spot ½ inch to the left of the sternal margin. It pierced the dilated conus arteriosus ¼ inch to the right of the middle line, and 1 inch below the pulmonary valve.
O, Superior vena cava.
P, Right branch of pulmonary artery.

Fig. 2.—The diseased cusps of the pulmonary valve.
Q points to the anterior cusp of the valve.

Plate II.

Fig. 3.—Shows the heart so dissected as to present to view the right ventricle, conus arteriosus, and pulmonary artery as one tube. A portion of the wall of the right ventricle, conus arteriosus, and pulmonary artery has been removed.

R points to the granular clot filling the saccular aneurism (G) on the left anterior wall of the pulmonary artery. A conical prolongation of this clot was found plugging the ductus arteriosus, and another prolongation of the clot was found partially obstructing the left branch of the pulmonary artery.

Fig I

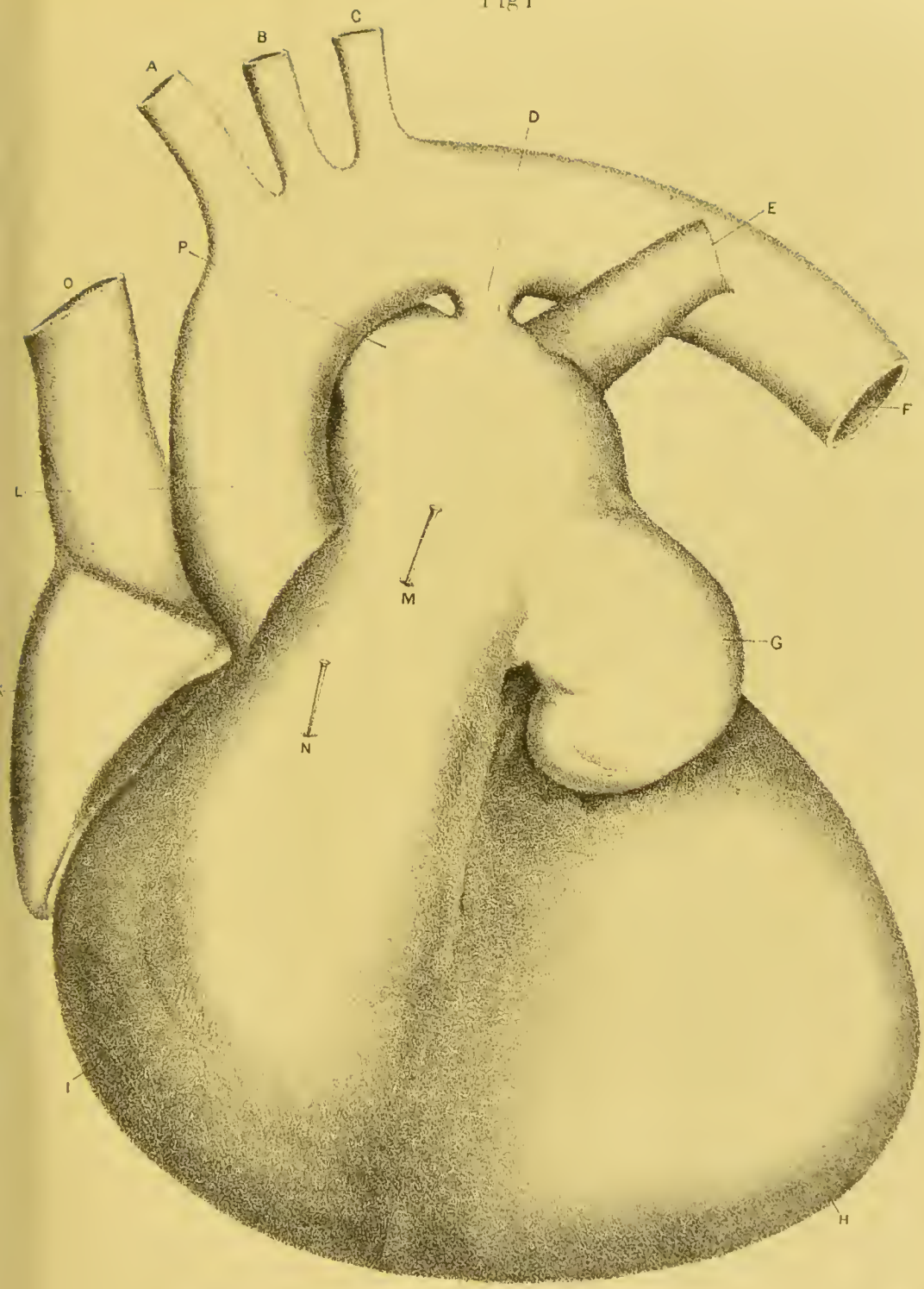


Fig II

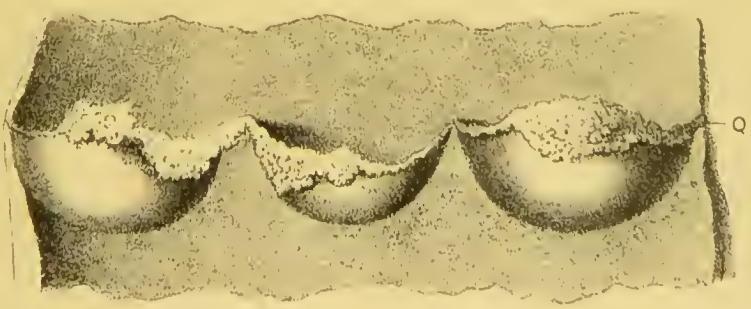


Fig III

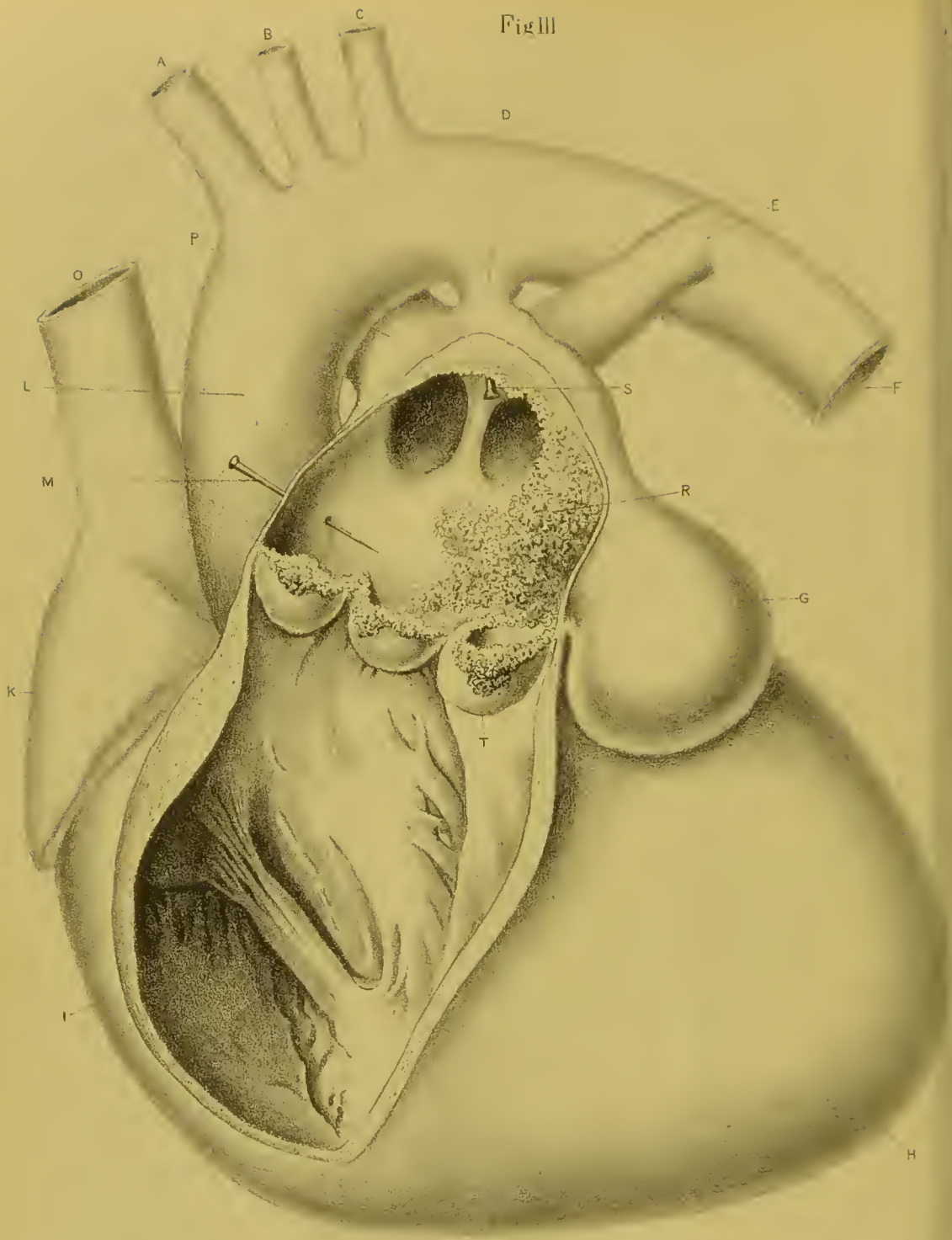
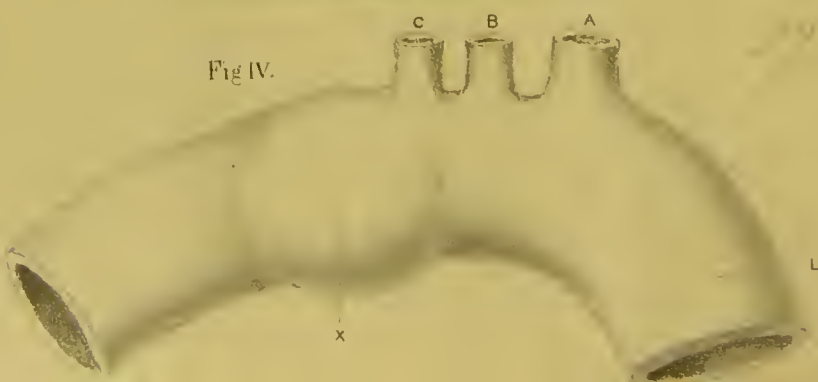


Fig IV.



S points to a large pin which was inserted into the lumen of the patent ductus arteriosus.

T points to the anterior cusp of the pulmonary valve.

FIG. 4.—Shows a portion of the ascending, transverse, and descending aorta, seen from behind.

× A distinct aneurismal bulging out, situated on the posterior wall of the aorta, just external to the line of the left subclavian artery, and immediately opposite to the aortic opening of the ductus arteriosus.

After hearing this description of the post-mortem examination, the first question which naturally suggests itself is, How were the murmurs which I have described produced? But before attempting to answer this question, let us shortly recapitulate the lesions which were found at the post-mortem examination in the heart and great bloodvessels. Let us look at these lesions following the course of the circulation of the blood.

The right auricle was greatly dilated. The tricuspid orifice was very slightly dilated, but as far as could be seen the tricuspid valve was competent. The right ventricle was much dilated and its wall thinned. The conus arteriosus was greatly dilated and its wall thinned. The pulmonary valve was very extensively diseased in its three cusps, each cusp being atrophied and shrunken, and covered over with granular vegetations on its ventricular side. The pulmonary artery itself was greatly dilated, and its left anterior wall converted into a saccular aneurism the size of a walnut, which was filled with granular clot. The ductus arteriosus once patent, allowing a goose quill to pass through it, was now partly plugged with clot. The left auricle was almost natural. The mitral valve was natural, except that its anterior cusp on the *auricular* surface presented a mass of vegetation the size of a small bean. The left ventricle was much dilated and the wall hypertrophied. The aortic valve was competent, but on its ventricular surface the anterior cusp presented a mass of dense vegetation the size of a small pea, while on the other cusps were small vegetations the size of pin heads.

Let us also remember that the needle No. 1, which was driven into the second left intercostal space at a spot one inch to the left of the sternal margin, pierced the pulmonary artery in its middle, just about ¼ inch above the diseased pulmonary valve, and that on stethoscopic examination of the heart, the murmurs referred to were heard with greatest intensity at this very spot.

Now, during the simultaneous contraction of the two ventricles, blood driven through the aortic valve and through the pulmonary valve would come in contact with cusps whose margins were rough with dendritic vegetations. In the case of the aortic valve it is certainly doubtful if a murmur was produced there, but in the case of the pulmonary valve, where the cusps were so extensively diseased, each cusp being shrunken and covered over on its ventricular surface with masses of vegetations, there can be little doubt,

I think, that a loud murmur was produced at that situation. If any murmur was produced at the aortic valve, I doubt if it was possible to hear it in the presence of the much louder murmur at the pulmonary valve. A careful examination of the aortic valve showed that it was competent, and that the pea-sized vegetation on the ventricular side of its anterior cusp would offer very little obstruction to the flow of blood through the valve. Hence I doubt if any murmur was produced at the aortic valve.

With regard to the mitral orifice,—if any murmur was produced there during the systole of the heart, it must have been a very feeble one, which could not be distinguished in the presence of the loud murmur at the pulmonary valve. The first loud murmur heard with such remarkable clearness in the pulmonary area could be very clearly heard at the apex of the heart.

I have already said that at the end of the first loud blowing and thrilling murmur a distinct click was heard. This click was clearly heard over the aortic area, and I believe was caused by the sudden closure of the aortic valve under great pressure. Did the blood rushing through the patent ductus arteriosus during the heart's systole contribute anything towards the first loud and thrilling murmur ? The ductus arteriosus is situated much deeper in the chest than the valve of the pulmonary artery, but it is likely that any murmur produced at that patent orifice would be most clearly heard at the spot where the pulmonary artery is nearest to the chest wall, *i.e.*, in the second intercostal space to the left of the sternal margin. It appears to me quite impossible that such a volume of blood as would be driven by the contraction of the right ventricle through the patent ductus arteriosus could pass through that orifice without distending it, and causing its margins to vibrate. That the blood was being driven through the patent ductus arteriosus with much force up to within three weeks of the girl's death was evident from the fact that there existed on the posterior wall of the aorta, just external to the left subclavian artery, an aneurismal dilatation or bulging out of that wall, which could have been only caused by a column of blood driven with much force against the wall in that situation ; and this bulging out of the posterior wall of the aorta was exactly opposite the aortic orifice of the patent ductus arteriosus. The ductus arteriosus in the fœtus is as large as the pulmonary artery itself, and I don't suppose that a murmur is produced by blood rushing through it during the fœtal circulation ; but when it is reduced to a tube which just admits a goose quill, and has a powerful right ventricle behind it, the quantity of blood which is driven through it must distend it and give rise to a vibratory or thrilling murmur at its margins.

With regard to the second murmur, which I have described as beginning immediately after the closure of the aortic valve, we have to look to the tricuspid orifice, to the mitral orifice, and to

the ductus arteriosus for the explanation of its production. Bearing in mind that the tricuspid orifice was healthy, and its valve competent, and that the aortic valve was competent, little or no regurgitation could take place through those valves. Now, when the two ventricles contracted the blood driven by the hypertrophied left ventricle would be sent with much force into the aorta, and at the same time, by the contraction of the right ventricle, a considerable quantity of blood would be driven through the patent ductus arteriosus, also into the aorta, and as a result that vessel would be greatly distended. According to the well-known law, it would recoil with the same force with which it had been distended. The aortic valve being competent, a certain quantity of blood would be driven back with much force into the pulmonary artery during the recoil of the aorta; and supposing the pulmonary valve was competent, a great distending force would be exerted, according to Pascal's law, upon the inner wall of the pulmonary artery and on the pulmonary valve; and if this valve was incompetent, then the distending force would be exerted on the inner wall of the conus arteriosus and of the right ventricle, as well as upon the inner wall of the pulmonary artery.

It appears to me that the aneurismal sac on the pulmonary artery was produced in one of the two following ways:—Either the recoil current through the ductus arteriosus, by distending the pulmonary artery in all directions, gradually caused that vessel to dilate and to give way at one part, say its weakest part, until it was bulged out in the form of a sac, or the recoil current impinging directly on one part of the wall of the pulmonary artery with much force, gradually bulged out that part until the aneurismal sac was produced. It is interesting to note that if the axis of the lumen of the ductus arteriosus was prolonged, at one end it would strike the middle of the saccular aneurism on the pulmonary artery, and at the other end would strike the middle of the aneurismal dilatation on the posterior wall of the aorta, which I have described. From which observation it is only fair to conclude that both these aneurismal dilatations were produced in the same way, namely, by the distending force of the to and fro current, driven through the patent ductus arteriosus, acting on the vessels at the spots where the aneurisms were produced. There can be no doubt that as a result of the diseased state and incompetency of the pulmonary valve, a very great distending force must have been exerted upon the pulmonary artery, the conus arteriosus, and the right ventricle at one and the same time—all these structures, as one tube, being greatly dilated.

From the fact that the left auricle was very slightly dilated, it is only fair to conclude that there was very little obstruction, if any, to the flow of blood through the mitral orifice during the auricular systole; and although a good-sized vegetation was found on the

auricular surface of the aortic cusp of the mitral valve, it was quite evident that there was no obstruction to the flow of blood through the valve, as the auriculo-ventricular orifice was dilated rather than constricted. I believe, therefore, that no murmur whatever was produced at the mitral orifice during the auricular contraction. We have then only the pulmonary artery and the patent ductus arteriosus to account for the production of the second murmur. As the result of careful observation, it was noted that the second murmur began immediately after the closure of the aortic valve, and was heard most distinctly in the second intercostal space to the left of the sternal margin. Was it produced by the blood rushing back through the ductus arteriosus during the powerful recoil of the aorta, or was it produced by regurgitation of blood through the diseased pulmonary valve? It was quite evident from a careful examination of the axis of the tube of the ductus arteriosus, and of the situation of the pulmonary valve, that the recoil current through the ductus arteriosus would impinge not on the cusps of that valve, but on a spot at least an inch away from them, outwardly towards the left, that spot being the centre of the saccular aneurism on the pulmonary artery. The rush of blood back through the patent ductus arteriosus mixing with the blood in the distended pulmonary artery, conus arteriosus, and right ventricle, would set up a commotion in that fluid, but I fail to see how the cusps of the diseased pulmonary valve could be set into a state of vibration sufficient to cause a murmur by the recoil current which would strike, not on them, but on the dilated wall of the pulmonary artery, fully an inch above them. It must be also borne in mind that the pulmonary artery at the situation of its valvular cusps was greatly dilated, its circumference being at this spot $3\frac{1}{2}$ inches. The incompetency of this valve was therefore marked in the highest degree. The post-mortem examination also showed that its cusps were much shrunken and atrophied, and that their ragged margins hung dependent into the ventricular cavity. These ragged edges would undoubtedly be thrown forward and caused to vibrate by the large volume of blood from the right ventricle rushing past them during the systole, after the closure of the right auriculo-ventricular valve. The fluid contents of the large right ventricle would have to be *squeezed* through the comparatively narrow gate of the pulmonary valve, and the ragged margins of that gate would vibrate in consequence. But it appears to me the case is quite different when we come to consider what took place during the rush of blood back through the ductus arteriosus. Granting that this recoil current did strike on the cusps of the pulmonary valve, in consequence of the *smallness* of its volume as compared with the great incompetency of the pulmonary valve, there would be very little obstruction to its regurgitant flow into the right ventricle. I therefore think that whatever regurgitant murmur was produced at the seat of the pulmonary valve was, comparatively

speaking, nothing in the presence of the loud murmur produced at the orifice of the ductus arteriosus during the recoil of the aorta.

It is quite clear that a large quantity of blood could not possibly be pumped back through the patent ductus arteriosus without causing that tube to dilate, and as a result the blood rushing past the distended margins of the tube would set up a vibration of these margins and thus give rise to a murmur.

I have therefore come to the conclusion that the first murmur really consisted of two murmurs, the one being produced by the rush of blood past the diseased cusps of the pulmonary valve, and the other being produced by the blood rushing through the distended ductus arteriosus, both together constituting the loud blowing and thrilling murmur which accompanied the systole of the ventricles. The second murmur, I believe, was produced by the blood rushing back through the patent ductus arteriosus during the recoil of the aorta.

It cannot have escaped your recollection that on the return of my patient from the country, on the 31st August 1882, she was in a state of great danger from extreme congestion of the lungs. The whole venous system was greatly engorged, and the urine was loaded with albumen. She had constant cough with frothy and bloody expectoration; she died within a month after her return. An examination of the lungs after death, especially the right lung, showed that for a considerable time previous to her death hæmorrhagic infarcti had been forming in those organs—the right lung throughout a great area being rendered useless in consequence. There can be no doubt that these infarcti had formed as the result of embolic plugging of the terminal branches of the pulmonary artery by little pieces of vegetations detached from the extensively diseased cusps of the pulmonary valve, or by little fragments of coagulum detached from the granular clot filling the aneurism on the pulmonary artery. The post-mortem examination also showed that the ductus arteriosus was almost completely plugged by a conical clot which projected from the clot in the saccular aneurism on the pulmonary artery, and that the large left branch of the pulmonary artery was greatly obstructed by a clot from the same source. Did these conditions exist when my patient returned from the country on the 31st of August, more than three weeks before her death? The history of the case shows that she began to be extremely ill a day or two before her return on the 31st August. When I saw her on that day her dyspnœa was most distressing. The venous system was everywhere terribly engorged; the veins of the neck being greatly distended. The urine contained a very large quantity of albumen, about one-fifth. There was crepitation in all parts of her lungs. She had constant cough with bloody and frothy expectoration; and from this time on to her death there were frequent attacks of hæmoptysis and epistaxis. The heart all the time was heaving against the wall of

the chest with a remarkably strong impulse, and, strange to say, the two loud murmurs had entirely disappeared; but during the violently wobbling action of the heart one could hear the two sounds "filupp," "flupp" quite distinctly. Her extremely distressing symptoms evidently began as the result of the plugging of the terminal branches of the pulmonary artery, and as the result of the obstruction in the left branch of the pulmonary artery, and the consequent obstruction to the circulation through the lungs.

How is it possible to account for the disappearance of the two loud murmurs and for the production of the sounds "filupp," "flupp?"

If my explanation of the production of the two loud murmurs is correct, I think their disappearance may be accounted for in the following way:—Bearing in mind that numerous terminal branches of the pulmonary artery in both lungs were plugged, and that one of the main branches of the pulmonary artery itself was greatly obstructed, and that both lungs were in a state of catarrhal pneumonia, and that the kidneys and spleen were greatly congested—the whole forming an impediment to the circulation of blood through the lungs which the distended and dilated right side of the heart could not overcome—it would necessarily follow in consequence of this engorged state of the whole right side of the heart, that too small a quantity of blood would be driven past the cusps of the pulmonary valve to set them in vibratory commotion. Hence the absence of the first part of the loud systolic murmur. But in addition, the almost complete closure of the ductus arteriosus by a conical clot from the aneurism would effectually prevent the passage of a sufficient quantity of blood through that opening during the heart's systole as would cause its margins to vibrate. Hence the disappearance of the second part of the loud systolic murmur. In the same way this plugging of the once patent ductus arteriosus would equally prevent the rush back of blood during the recoil of the aorta. Hence the disappearance of the second loud murmur.

With regard to the production of the sounds "filupp," "flupp," heard over the base of the heart, it was quite evident that the first was caused by the reduplication and accentuation of the first sound of the heart with the systole, and that the second shorter sound was caused by the rapid closure of the aortic valve—the whole arterial vascular system being in a state of great tension due to the block to the circulation in the right side of the heart and in the lungs.

Gentlemen, after hearing this paper so far you will ask, Have we learnt anything, either from the symptoms observed during life or from the post-mortem appearances, which would enable us in the future accurately to diagnose a case of patent ductus arteriosus with aneurism of the pulmonary artery?

The chief facts in connexion with the physical symptoms observed during life which I have brought before you are these:—

On auscultation a loud, blowing, thrilling murmur accompanying the ventricular systole, was heard with maximum intensity in the second left intercostal space, fully an inch to the left of the sternum, and at the same time a strong heaving pulsation was communicated to the stethoscope at this spot. The best anatomical authorities state that the left border of the pulmonary artery lies in the second intercostal space, at a distance of three-fourths of an inch to the left of the margin of the sternum. If we meet with a case in which a set of symptoms, such as those just described, exist, we may fairly conclude that there is a considerable dilatation of the pulmonary artery present. A dilated pulmonary artery alone would not, however, give rise to such an extraordinary murmur as was heard with the ventricular systole in my patient's case. Dilatation of the pulmonary artery above its valve is such a rare thing in a young person, that we must look for some special cause of obstruction *in front* of the dilatation to account for it. In connexion with a dilated pulmonary artery a murmur may or may not be present. Its presence with the ventricular systole will depend on whether the pulmonary valve is diseased or not, or whether there is some obstruction to the flow of blood through the pulmonary artery at a point farther on than the seat of the pulmonary valve. So rare is the existence of a dilated pulmonary artery in a young person that if we find an example of it we may suspect that a patent ductus arteriosus is the cause of it, because such an obstruction in the lungs or in the pulmonary artery itself, apart from a patent ductus arteriosus, as would cause a great dilatation of the pulmonary artery, is almost unknown.

An acquaintance with Pascal's law of the diffusion of pressure in fluids will at once enable us to understand how even a strongly walled pulmonary artery may gradually become dilated into a saccular aneurism, if there is a leak back from the aorta into the pulmonary artery, as exists in the case of a patent ductus arteriosus. The short wide tube of the pulmonary artery above its valve is exposed to a very great extensile pressure by the column of blood escaping back through that patent orifice acting on the fluid contents of that artery. Dilatation once begun, the extensile pressure gradually becomes greater and greater, until the tube is burst or a saccular aneurism is produced. But not only is this pressure exerted on the inner wall of the pulmonary artery, but the cusps of the pulmonary valve are exposed to similar pressure. Sooner or later they give way, and become diseased and incompetent.

There can be no doubt, then, that by far the commonest cause of an aneurismal dilatation of the pulmonary artery is the existence of a patent ductus arteriosus. When this exists, a loud thrilling

murmur will be heard accompanying the ventricular systole caused by the rush of blood through that orifice into the aorta, and in the same way after the closure of the aortic valve, another murmur would be caused by the rush of blood back through that orifice during the recoil of the aorta, and this rush back current is the one which gradually causes the dilatation of the pulmonary artery.

If, then, we meet with a case in which there is no history of lung disease such as would cause obstruction to the flow of the circulation through the lungs, and if on auscultation in the second left intercostal space we find there is a heaving pulsation communicated to the stethoscope with the ventricular systole, and at the same time a loud thrilling murmur is heard with maximum intensity at this very spot, and also heard in lines radiating upwards and outwards from this spot, and if this murmur is followed by another, similar in character, immediately following the closure of the aortic valve, we may fairly conclude that the cause of the aneurismal dilatation of the pulmonary artery, and of the two thrilling murmurs, is a patent ductus arteriosus.

These murmurs and the dilatation of the pulmonary artery may exist without any co-existing disease of the pulmonary valve. In fact, I should think that freedom from disease and competency of that valve was a necessary condition for the production of any marked aneurismal dilatation of the pulmonary artery itself. No doubt the valve must sooner or later become incompetent, and then the whole right side of the heart becomes subjected to great extensile pressure, and dilatation results, as was so well observed in the right heart of my patient.

I do not believe that the attack of scarlet fever from which my patient suffered six or seven years before she came to consult me had in any way affected the right side of her heart. She was able to do hard work, even to dance, up to within a comparatively short time before I first saw her. We know that even a slight cold in such a case would be sufficient to throw much pressure on the pulmonary valve, and my belief is that her pulmonary valve began to give way, and became crumpled and diseased as soon as embolic plugging of the terminal branches of the pulmonary artery took place.

From a careful examination of the aneurism and of the pulmonary valve, I think there can be no doubt that the former existed a long time before the latter began to be diseased and incompetent, and it was only when the latter became so that the distressing symptoms commenced which ultimately carried off my patient.

The mere presence of a patent ductus arteriosus and the consequent intermingling of venous and arterial blood does not seem to act very injuriously on the health of the patient. In my patient's case there was very little cyanosis at the first, and all

her distressing symptoms resulted from the extreme dilatation of the whole right side of the heart, and the obstruction to the flow of blood through the lungs.

With regard to the literature of this subject there is very little to quote from or refer to. As far as I have been able to ascertain, there is no case recorded in this country in which a post-mortem examination has brought to light the pathological conditions which have been described in this paper.

In the *Glasgow Medical Journal* for 1879, Dr Wood Smith of Glasgow has a short paper, with a woodcut, on "A Probable Case of Aneurism of the Pulmonary Artery and Ductus Arteriosus," but the case was by no means clearly made out. The patient is still alive and in good health. In vol. xvii., *Archiv der Heilkunde*, Dr Lüttich has a case entitled "Fall von Aneurysma des Ductus Bottali und Thrombose der Aorta," and in Rokitansky's *Krankheiten der Arterien* there are several illustrations of cases of patent ductus arteriosus, but none of them resemble the case I have brought before you this evening.

I cannot close this paper without expressing my deep gratitude to Dr Wyllie for the careful manner in which he performed the post-mortem examination in this case, and for his accurate description of the pathological conditions found in connexion with the heart and large bloodvessels, and I am the first to admit that the value of this paper consists in the scientific accuracy of his descriptions.

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